BLOOD LEAD LEVELS IN THE ELDERLY

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Introduction

Lead is ubiquitious in the environment and human tissues. Mobile and stationary sources of anthropogenic lead tend to be concentrated in areas of high population density. At concentrations previously regarded as 'safe', lead has been implicated in causing neurobehavioural impairment (Needleman et al., 1990), hearing deficits (Robinson et al., 1985), growth retardation (Schwartz et al., 1986), inhibition of haem biosynthesis (EPA, 1986), hypertension (Harlan, 1985) and suppression of the immune response (Hemphill et al., 1971).

In 1983, the joint WHO/UNEP committee found median values of blood lead concentrations (PbB) of 247μg/L in middle-aged non smoking Maltese males (Braux et al., 1985). The study suggested that the main source of lead exposure was food, especially pasta and certain vegetables. The maximum median value proposed by the EEC is 200μg/L (Council Directive 1977).

In all industrialised nations, the 85-plus age group is the fastest growing group. Unfortunately, toxicology has no proven methodology to predict the possible interaction with aging of low level exposure to many chemicals in the environment (Williams et al., 1987).

This study consists of the analysis of PbB in an elderly population. Variables which may affect PbB were collected via patient medical records and a patient interview.

Methodology

(a) Analysis of PbB

Venous blood samples were obtained from 92 elderly non-anaemic subjects (age range: 51-92 years) located at the St Vincent de Paule Residence for the Elderly (S.V.P.R.), transferred into evacuated heparinised tubes and stored at 4°C. Time of sampling to time of analysis never exceeded one week. All glassware was acid-washed and the lead content of the reagents verified. Blood samples were digested in duplicate by a perchloric/sulphuric acid mixture (wet-ashing). The samples were diluted and analysed by DPASV using the Metrohm Model 646 VA Processor.

(b) Survey

Each subject's medical record was searched for data concerning age, sex, previous residence, date of admission into residence, underlying disease (hypertension, diabetes mellitus, and disorders which were categorized into cardiac, neurological, osteological and renal), and current medication.

Subjects were interviewed to obtain data on past occupation, past reactional activities, smoking habits, alcohol intake, number of children (asked to females only), and awareness on the health implications of lead.

Results

A two-tailed P value of 0.05 was chosen to determine statistical significance. Table 1 shows the mean, standard deviation and range of PbB and age for the 92 subjects. The univariate distribution of PbB is shown in Fig. 1. PbB were correlated with a number of independent variables by linear regression analyses (Table 2). PbB were also correlated with the social characteristics of each patient using t-tests and/or analysis of variance (Table 3).

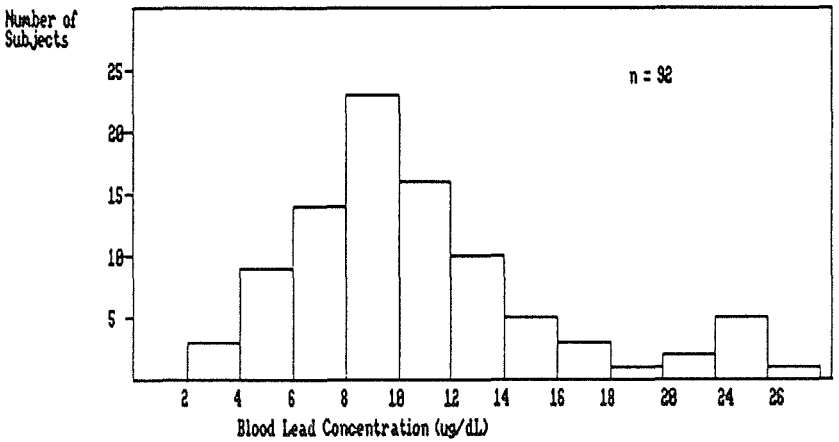

Fig 1. Distribution of blood lead concentrations in 92 elderly subjects.

Table 1: Mean, standard deviation and range of PbB and age of 92 elderly subjects

Variable	All (n=92)			Males (n=26)		Females (n=66)	
	Mean	SD	Range	Mean	SD	Mean	SD
PbB (µg/dL)	10.70	5.07	3.26-25.71	11.07	5.70	10.56	4.84
Age (years)	77.66	9.93	51-92	76.53	10.95	78.10	9.55

SD = standard deviation

PbB was found to be increased in hypertensives and patients suffering from renal or cardiac impairment. It was decreased in patients with neurological or osteological disorders. None of these differences were significant, however. PbB for diabetics and non-diabetics were almost similar.

Table 2: Correlation of PbB with age, previous residence, duration of stay in S.V.P.R. and number of children in 92 elderly subjects

Variable	R	P
age	0.137	0.190
age^2	0.132	0.208
location*	0.019	0.860
duration of stay	-0.106	0.316
number of children	0.211	0.103(f)

f = females only
* = according to voting population

Table 3: Correlation of PbB with the social characteristics of 92 elderly subjects

Past residence	resid/indus*	heavy traffic	background
	8.67	11.04	11.65
Past occupation	housebound	non-lead-prone	lead-prone
	10.83	9.77	12.25
Past recreational	indoor	outdoor	
activities	9.77	12.75	
Smoking	non-smoker	ex-smoker	smoker
	10.50	12.40	9.79
Alcohol	non-drinker	ex-occ.drinker	ex-freq.drinker
	10.09	13.09	15.86

* residential/industrial

Only 23% of the 80 patients interviewed were aware of the health risks afforded by lead.

Discussion

The blood lead concentration is generally considered as a measure of the biologically active metal in the body and reflects environmental exposure of an individual (Sartor et al.,1980). The mean PbB in this study, 10.7µg/dL was of the same order of magnitude as in most Western populations not exposed to lead at work (Elwood et al, 1988).

The PbB are substantially lower than those measured in 1983. This may be due to:

- different analytical techniques (AAS vs DPASV)
- the downward trend in measurable PbB observed by several European workers (Drasch et al., 1987)
- a reduction in exposures to atmospheric lead regarding this elderly population
- altered toxicokinetics in the elderly, such as a decrease in the rate of lead uptake from the gut (Quartermann et al., 1978).

Lack of any sex-related significant differences in PbB may be due to the fact that this difference is in part due to a higher potential for

occupational exposure in men (Mahaffey et al., 1982); in this population, both sexes are subjected to the same environmental influences.

Hypertensives were found to have higher PbB than normotensives; however this did not reach statistical significance. The increase in PbB may parallel the increase in blood pressure until some limit value, so that such a trend is apparent only when factors such as age do not competitively increase blood pressure by greater amounts.

The previous social characteristics of the subjects, such as past occupation and previous residence do not appear to have affected PbB. This is in concordance with the fact that PbB is an indicator of **current** environmental exposure. Outdoor recreational activities contributed to a higher PbB than indoor activities ($P=0.019$). This was attributed to atmospheric lead exposure via traffic in view of the Maltese custom of staying outside the front door chatting with people, a practice common among the elderly.

The study also showed higher PbB for ex-drinkers when compared to subjects who never drank ($P<0.001$). This was probably due to impaired excretion of lead or release from lead stores in cirrhotic livers. The lead contribution from cigarette smoking was not found to be great enough to increase the PbB above that found in non smokers.

Conclusion

1. The low PbB obtained warrants another major lead screening program, comprising all age groups. The problem of choosing the right analytical technique is extremely important.

2. Moderate to high alcohol consumers are at a greater risk from the subclinical effects of lead.

3. Further analysis is required to detect age-related dispositional changes regarding environmental toxins and the elderly.

References

Braux P. et al. Assessment of Human Exposure to Lead: Comparison between Belgium, Malta, Mexico and Sweden. Braux P., Svantengren M., Eds. UNEP/WHO 1985.

Council Directive of 29 March 1977 on biological screening of the population for lead (77/312/EEC) Off J Eur Comm 1977; No L 105/10-17.

Drasch G.A. et al. Lead in human bones. Investigation on an occupationally non-exposed population in southern Bavaria (FRG) 1. Adult. *Sci Total Environ* 1987; 647: 303-315.

Elwood P.C. et al. Blood pressure and blood lead in surveys in Wales. Am.J.Epidem. 1988; 127: 942-945.

EPA (Environmental Protection Agency) 1986. Air quality criteria for lead. Air quality criteria for lead. June 1986 and Addendum, September 1986. Research Triangle Park, N.C. : Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, EPA. EPA 600/8-83-018F.

Harlan L.C. Blood lead and blood pressure. *JAMA* 1985; 253: 530-534.

Hemphill F.E., Kaeberle M.L., Buck W.B. 1971. Lead suppression of mouse resistance to *Salmonalla typhimurium*. Science 1972; 1031-1032.

Mahaffey P.R. et al. National estimates of blood lead levels: United States, 1976-1980. *NEJM* 1982; 307: 573-579.

Needleman H.L., Gatsonis C.A. Low level lead exposure and the I.Q. of children - A meta-analysis of modern studies. *JAMA* 1990; 263: 673-678.

Quartermann J., Morrison E. The effect of age on the absorption and excretion of lead. *Env. Res.* 1978; 17: 178-183.

Robinson G., Baumann S., Kleinbaum D. 1985. Effects of low to moderate lead exposure on brainstem auditory evoked potentials in children. Copenhagen, Denmark: WHO Regional Office for Europe, 177-182 (Environmental Health Document 3).

Sartor F., Rondia D. Blood lead levels and age: a study in two male urban populations not occupationally exposed. *Arch Environ Health* 1980; 35: 110-116.

Schwartz J., Angle C., Pitcher H. Relationship between childhood lead levels and stature. *Pediatrics* 1986; 77: 281-288.

Williams J.R. et al. 1987. Interactions of Aging and Environmental Agents: *The Toxicological Perspective. In: Environmental Toxicity and the Aging Process*: Proceedings of a Workshop held in Colombus, Maryland (Baker S.R. and Rogul M. Eds.) pp 81-135. A.R. Liss, New York.